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| #5     | Search inhibitor and nvl           | 10:34:27 | 2      |
| #4     | Search inhibtor and NVL            | 10:34:25 | 0      |
| #3     | Search Marionneau S 2002           | 09:56:29 | 3      |
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| #1     | Search Severine M 2002             | 09:55:57 | 0      |

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DATE: Monday, October 29, 2007

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| <input type="checkbox"/> | L9       | (Fug-apha-2)  | 0         |
|                          |          | <i>DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>                               |           |
| <input type="checkbox"/> | L8       | L7  | 11        |
|                          |          | <i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>      |           |
| <input type="checkbox"/> | L7       | oligosaccharide and L5  | 52        |
|                          |          | <i>DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>                               |           |
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|                          |          | <i>DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>           |           |
| <input type="checkbox"/> | L5       | L3 and composition  | 52        |
| <input type="checkbox"/> | L4       | L2 and l3   | 0         |
| <input type="checkbox"/> | L3       | (GalNAc-alpha-2)  | 52        |
| <input type="checkbox"/> | L2       | histo adj blood   | 120       |
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| 10:34:27 | <u>2</u> |
| 10:34:25 | <u>0</u> |
| 09:56:29 | <u>3</u> |
| 09:55:59 | <u>1</u> |
| 09:55:57 | <u>0</u> |

#6 Search inhibitor and Norwalk virus

#5 Search inhibitor and nvl

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#3 Search Marionneau S 2002

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1 OLIGOSACCHARID  
2 OLIGOSACCHARIDS  
L1 3 OLIGOSACCHARID  
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=> "Fuc-alpha-2"

1865 "FUC"  
5 "FUCS"  
1870 "FUC"  
( "FUC" OR "FUCS" )  
1720798 "ALPHA"  
2480 "ALPHAS"  
1720907 "ALPHA"  
( "ALPHA" OR "ALPHAS" )  
9348277 "2"  
L2 15 "FUC-ALPHA-2"  
( "FUC" (W) "ALPHA" (W) "2" )

=> norwalk and L2

631 NORWALK  
L3 0 NORWALK AND L2

=> inhibitor and L2

555233 INHIBITOR  
558378 INHIBITORS  
871400 INHIBITOR  
(INHIBITOR OR INHIBITORS)  
L4 2 INHIBITOR AND L2

=> D L4 IBIB ABS 1-2

L4 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1997:375443 CAPLUS

DOCUMENT NUMBER: 127:62243

TITLE: High affinity binding of the Entamoeba histolytica lectin to polyvalent N-acetylgalactosaminides

AUTHOR(S): Schnaar, Ronald L.; Adler, Pablo; Lee, Yuan C.; Lee, Reiko T.; Petri, William A., Jr.

CORPORATE SOURCE: Johns Hopkins University, Baltimore, MD, USA

SOURCE: Proceedings of the ERDEC Scientific Conference on Chemical and Biological Defense Research, Aberdeen Proving Ground, Md., Nov. 15-18, 1994 (1996), Meeting

Date 1994, 511-517. Editor(s): Berg, Dorothy A.  
National Technical Information Service: Springfield,  
Va.

CODEN: 64NAAX

DOCUMENT TYPE:

Conference

LANGUAGE:

English

AB Entamoeba histolytica trophozoites initiate pathogenic colonization by adherence to host colonic epithelial glycoconjugates via an amoebic surface lectin which binds to non-reducing terminal galactose (Gal) and N-acetylgalactosamine (GalNAc) residues. A series of natural and synthetic monovalent and multivalent carbohydrate ligands was screened for inhibition of E. histolytica lectin-mediated human red cell hemagglutination. This screen revealed that: (i) the synthetic multivalent neoglycoprotein GalNAc39BSA (having an average of 39 GalNAc residues linked to lysines on bovine serum albumin) is among the most potent ligands tested, with an affinity 140,000-fold higher than monovalent GalNAc and 500,000-fold higher than monovalent Gal; and (ii) small synthetic multivalent ligands which bind with high affinity to the mammalian hepatic lectin (which has similar monosaccharide specificity) do not bind with high affinity to the E. histolytica lectin, revealing a distinct difference in preferred spacing of carbohydrate determinants for binding to the two lectins. The high affinity of GalNAc39BSA allowed facile radioligand binding studies, revealing saturable binding of <sup>125</sup>I-GalNAc39BSA to E. histolytica membranes ( $K_D = 10 \pm 3$  nM,  $B_{max} = 0.9 \pm 0.08$  pmol/mg membrane protein). Maximal E. histolytica lectin binding required either the presence of a low concentration of calcium chloride (300  $\mu$ M) or a high concentration (50 mM) of sodium chloride, and had a broad pH maximum (pH 6-9). GalNAc was 7-fold more potent than Gal in blocking radioligand binding, while Gal39BSA was 160-fold more potent than Gal40BSA. The presence of a hydrophobic aglycon (p-nitrophenyl  $\beta$ -N-acetylgalactosaminide) enhanced affinity 8-fold compared to the free monosaccharide, and the  $\beta$  glycoside was a 2-fold better inhibitor than the  $\alpha$  glycoside. When synthetic polyvalent saccharide-derivatized linear polymers were tested as inhibitors, the (GalNAc $\beta$ ) and (GalNAc $\alpha$ 3Gal $\beta$ ) derivs. were the most potent, with (GalNAc $\alpha$ ) and (GalNAc $\alpha$ 3( Fuc.  $\alpha$ .2)Gal $\beta$ ) derivs. much weaker inhibitors. The data support a model in which a unique pattern of spaced multiple GalNAc residues are the highest affinity targets for the E. histolytica lectin.

L4 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1995:440655 CAPLUS

DOCUMENT NUMBER: 123:3738

TITLE: High affinity binding of the Entamoeba histolytica lectin to polyvalent N-acetylgalactosaminides

AUTHOR(S): Adler, Pablo; Woods, Sheila J.; Lee, Yuan C.; Lee, Reiko T.; Petri, William A., Jr.; Schnaar, Ronald L.

CORPORATE SOURCE: Department Pharmacology Molecular Science, Johns Hopkins School Medicine, Baltimore, MD, 21205, USA

SOURCE: Journal of Biological Chemistry (1995), 270(10), 5164-71

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Entamoeba histolytica trophozoites initiate pathogenic colonization by adherence to host glycoconjugates via an amoebic surface lectin which binds to galactose (Gal) and N-acetylgalactosamine (GalNAc) residues. Monovalent and multivalent carbohydrate ligands were screened for inhibition of E. histolytica lectin-mediated human red cell hemagglutination, revealing that: (i) the synthetic multivalent neoglycoprotein GalNAc39BSA (having an average of 39 GalNAc residues linked to

bovine serum albumin) was 140,000-fold more potent an inhibitor than monovalent GalNAc and 500,000-fold more potent than monovalent Gal; and (ii) small synthetic multivalent ligands which bind with high affinity to the mammalian hepatic Gal/GalNAc lectin do not bind with high affinity to the *E. histolytica* lectin. Radioligand binding studies revealed saturable binding of <sup>125</sup>I-GalNAc39BSA to *E. histolytica* membranes (K<sub>D</sub> = 10 nM, B<sub>max</sub> = 0.9 pmol/mg membrane protein). Maximal binding required the presence of calcium chloride (300 μM) or sodium chloride (50 mM), and had a broad pH maximum (pH 6-9). GalNAc39BSA was 200,000-fold more potent than monovalent GalNAc in blocking radio-ligand binding. Among synthetic saccharide-derivatized linear polymers, the GalNAcβ and GalNAcα3Galβ derivs. were the most potent, with GalNAcα and GalNAcα3( Fuc.alpha.2)Galβ derivs. much weaker. The data support a model in which a unique pattern of spaced multiple GalNAc residues are the highest affinity targets for the *E. histolytica* lectin.

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